# The Electroencephalogram in Pediatrics

## The Indications for Use and the Limitations

MERL CARSON, *M.D.*, and RICHARD D. WALTER, *M.D.*, Los Angeles

• **Electroencephalograms normally undergo pronounced serial changes during maturation of the brain.**

**Many physiological and nonphysiological influences may alter the EEG tracing.**

**As regards EEG's, appraisal of cerebral function is based on comparison with normal standards of frequency, rhythm, wave configuration and amplitude, bilateral synchrony, and response to various physiological stimuli.**

**Various abnormal EEG patterns are fairly constantly related to concomitant clinical disease.**

**A laboratory tool which may be used to supplement clinical data but not to make a clinical diagnosis, the EEG may also sometimes be of value in helping to suggest prognosis and in helping with evaluation of a therapeutic program.**

ELECTROENCEPHALOGRAPHY, the study of electrical activity of the brain, has been made possible by development of electronic machines which amplify and record this activity upon a moving chart. The activity in numerous parts of the head is simultaneously tested through individual amplifiers, and from these recordings an accurate appraisal of departures from norms can be made. Electrical activity recorded in any one amplifier (channel) does not represent total discharge from a single underlying segment of brain, but represents the difference in electrical potential between two areas underlying two connecting electrodes.

Cellular physiologists have concluded that periodic electrical discharges are a function of all living cells. The exact mechanism may not be entirely elucidated but a difference between intra- and extra-cellular potential is apparently a function of the semipermeable cell membrane and ionic transfer. It is likely that these discharges are the by-product of cellular metabolism and ordinarily have no specific function. The origin of these discharges in the central nervous system was formerly thought to be the axons of nerve cells. New histochemical evidence suggests instead that they may originate in apical dendrites of the third cortical layer.

Serial electroencephalograms normally undergo pronounced changes during the period of anatomical and functional development of the brain. It is postulated that the earliest cortical, electrical activity appears when adequate numbers of nerve cells have developed there, even before definitive interneuronal connections are established. Then, as the number of cells increases, there is increase in amplitude with continued lower frequency waves; and at last as myelination and functional connections become complete, the wave amplitude decreases and the frequency increases, developing into the mature, adult wave pattern.

Studies by Lindsley, Henry, Smith, Gibbs and others have clarified details of the normal maturing electroencephalogram (EEG) of children. At birth, irregular, slow 0.5-3.5 cps. (cycles per second) waves are present over the cortex with accentuated activity over the parietal areas. At six months there is high voltage, 4-6 cps. activity maximal over temporal and occipital areas, which increases to 5-8 cps. by one year of age, especially evident over the occipital area. By four to five years there is a steady 7-8 cps. activity in the occipital area, which increases to the adult 9-10 cps. wave pattern by nine years. At this age, however, an excess of slow 5-7 cps. waves is still present over the parietal and temporal areas. Typical adult patterns are frequently seen at age 12 years and by 19 years almost all persons have adult configurations.

*Physiological Variables.* The normal EEG may be influenced by many physiological variables,[12,15] some of which are incorporated in the routine study of each patient. Drowsiness and sleep profoundly affect the EEG, causing slowing and increased amplitude of waves. This pattern may appear in short runs and be confused with seizure discharges. High voltage, spike-like 3-8 cps. waves, termed *biparietal humps* are common over frontal and parietal areas up to nine years of age. With increasing age they tend to become smaller and are more confined to the parietal areas. Sleep spindles of 12-14 cps. are evidence of light sleep and tend to disappear with deeper sleep. Very deep sleep is characterized by generalized slow 0.5 to 2.0 cps. waves. Hyperventilation for a standard period of three minutes re-

From the Division of Neurology, U.C.L.A. Medical Center, Los Angeles 24.

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sults in the reduction of carbon dioxide tension in blood and tissue fluids. Cortical pH increases and cortical electrical discharges become greater in amplitude and slower in frequency. Certain neurological disorders may be symptomatically induced by this cortical stimulation, such as initiation of convulsive phenomena in patients with petit mal epilepsy. Oxygen and glucose constitute the main energy sources of the brain. A decrease of 30 per cent in internal jugular vein blood oxygen tension results in a sudden shift of cortical activity to slower frequencies. Glucose oxidation accounts for over 90 per cent of brain cell respiration and a deficiency thereof results in a steady lowering of cortical electrical activity. Light-stimulated epileptic seizures have long been recognized and Berger first noticed alteration of normal EEG rhythm by photic stimulation of the retina.

It is common for clinicians to be doubtful regarding proper indications for an EEG examination. The procedure is valuable in providing a safe, easy method of evaluating functioning cerebral activity. Therefore, it can be helpful in evaluating patients with convulsive disorders or other clinical conditions that suggest cerebral functional abnormality. It is a supplement to clinical observation, not a substitute for it. As a laboratory procedure it is subject to various limitations and artifacts.

Evidences of cerebral malfunction are necessarily somewhat limited by the mechanical, two dimensional tracings. Tracings are adjudged normal because of conformance with standards of the frequency of rhythm, wave configuration and amplitude, bilateral synchrony, and response to various physiological stimuli such as hyperventilation, sleep and photic stimulation. Variation in these components is due to alteration of cellular electrical activity caused by altered intracellular metabolism. Although this metabolic abnormality may be due to a wide variety of causes, there is so little difference among them insofar as EEG activity is concerned that making fine etiological classification of abnormalities is exceedingly difficult or impossible, except in a certain few instances. This is particularly evident in patients with seizures that are secondary to anatomic cerebral abnormality (symptomatic epilepsy).

### SYMPTOMATIC EPILEPSY

The abnormalities of symptomatic epilepsy may be derived from widely diverse causes. Birth trauma and anoxia, inflammatory diseases of the brain and meninges, and abnormalities of development are the most common factors in early infancy and childhood. In older children metabolic disorders and trauma assume greater importance. The brain damage may be diffuse or localized, mild or severe. In severe cases considerable neuronal destruction may result in an area or areas of electrical silence. Nearby cortex may contain normally functioning neurons with interposed cortical neurons damaged and functioning in an abnormal manner. Discharges from this abnormally functioning area cause the periodic seizures seen in patients with symptomatic epilepsy. Diffuse cerebral damage is usually reflected by varying seizure patterns such as generalized, myoclonic massive spasms.

The great majority of clinical seizures are referable to anatomic cerebral damage. Etiological factors responsible for the damage may be grouped into several large categories.

1. *Physical and Toxic:* (Trauma and anoxia); birth injury; anoxia; closed head injuries; cerebral vascular occlusion; poisoning (lead, thallium, barbiturate, etc.); subdural hematoma.

2. *Inflammatory:* Viral, bacterial, and parasitic meningoencephalitis; brain abscess; postimmunization encephalitides; acute cerebellar ataxia; inclusion body encephalitis.

3. *Congenital and Developmental Malformations:* Sturge-Weber syndrome; mongolism; microcephaly; hydrocephalus; tuberous sclerosis; abnormalities of gyri and cerebral cortex.

4. *Metabolic and Degenerative Disorders:* Disease of cerebral white matter, (Schilder's disease, cerebral sclerosis of Krabbe, etc.); disease of cerebral grey matter (Tay-Sachs disease, Niemann-Pick disease); phenylpyruvic oligophrenia; thyroid disease.

5. *Intracranial Space-Occupying Mass:* Tumor; cyst.

Although the EEG in some of the above conditions may suggest specific etiologic factors, in most it will show a nonspecific response to cerebral damage. When the damage is moderate and diffuse, there is an initial slowing of frequency and increase in amplitude. If hyperventilation can be carried out, the response will likely be exaggerated with bursts of diffusely symmetrical, slower and higher waves. Progressively severe damage will be reflected by diffusely slower waves, 0.5 to 3 cps. with amplitude and frequency decreasing with increasing cerebral impairment, the recording becoming completely flat in patients with the most severe damage. Response of this type is obtained in widely diverse problems—as in acute head injury,[4,16] inflammatory meningoencephalitides,[18,19,22] in anoxia and poisonings—and is truly nonspecific. Patient recovery is attended by reversal of the above pattern, and if permanent damage has not occurred the normal EEG pattern for the chronological age will emerge.

In cases of permanent damage, persistent, slow waves and hyperventilation lability may be seen.

Certain conditions warrant further elaboration because of specific individual features. In infants with subdural hematoma or effusion the EEG is less helpful than in adults.[25,28] Various abnormalities have been described, such as focal 0.5 to 3 cps. waves or flattening over the affected side, generalized diffuse slowing, suppression of normal occipital alpha rhythm, and occasionally normal tracings.

Studies in cerebral palsy[1] indicate a correlation between abnormality of the EEG and seriousness of brain damage. Abnormal tracings were found by Perlstein and coworkers[23] in 90 per cent of a group of 212 such patients with accompanying convulsive seizures, and in only 44 per cent of those without seizures. They are most common in patients with spasticity and less common in those with ataxia or athetosis. The 4 to 6 year age group had the greatest number of abnormal EEG's, with spike waves in 77 per cent and petit mal discharges in 1 per cent.

Dawson's inclusion body encephalitis is a chronic, progressive illness, usually beginning in young childhood. The onset is insidious with gradual, progressive loss of intellectual achievements, increasing irritability, followed by increasing stupor, coma and death. EEG studies[7] demonstrate progressive disappearance of normal rhythms with appearance of episodes of high voltage, slow waves (1.5 to 3 cps.) which become periodic, approximately at 8-second intervals. These are interspersed with pronounced flattening of activity. Cobb and coworkers considered that order of encephalographic changes as characteristic of the disease, but Lesse and coworkers[18] described similar observations in encephalitis of other types, diffuse vascular disease (one patient) and congenital cerebral anomaly (one patient). One cannot then state this EEG complex is pathognomonic of Dawson's encephalitis, although its appearance strongly suggests this diagnosis.

Microcephaly has been associated with abnormal EEG's in 73 per cent of a study group[10] with direct relationship between clinical severity of disease and degree of EEG abnormality. Many kinds of electrical abnormalities were found but the most striking feature was presence of decreased amplitude in 63 per cent of cases. In hydrocephalus the EEG abnormalities are similarly varied, with flattening present in the more severe cases.[9]

Degenerative diseases of the cerebral cortex are characterized by early disruption of the normal rhythm[6,8] by slower waves. There is progressive disorganization with generalized runs of 2 cps. waves accompanied by irregularly appearing, nonfocal spike waves. Visible myoclonic jerks frequently accompany the EEG spikes.

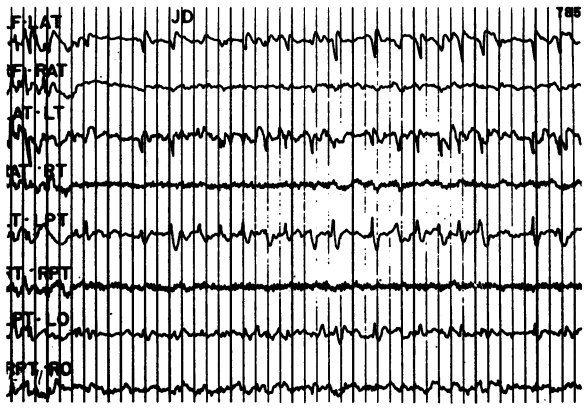


**Figure 1.—Localized spike waves with phase reversal over left temporal area.**

Space-occupying intracerebral masses may cause electrical abnormality.[3] Static, noninflammatory cysts or tumors near the cortex will usually cause some localized slowing, but if they be deep there may be no manifest EEG abnormality. Subtentorial tumors are more common in pediatrics and commonly cause bilaterally synchronous, high voltage slow waves with no focal or paroxysmal features.

Psychomotor or temporal lobe seizures are characterized by bizarre patterns of abnormal and inappropriate behavior.[21] These commonly occur as repetitious, semipurposeful movements, performed without the patient being conscious of the event. They may be of only a few seconds' duration, or may persist for hours. The termination of the seizure is usually marked by confusion and amnesia for the entire episode. The EEG concomitants may be quite variable. Gibbs emphasized the importance of spike foci over the anterior temporal area (Figure 1). The waking EEG is frequently normal, sleep being necessary to activate the electrical abnormality.

Infantile spasms are usually associated with a characteristic EEG pattern known as *Hypsarhythmia.*[5] Children with this condition frequently appear to develop normally until onset of seizures, usually during the first year of life. Mental impairment becomes evident with increasing frequency and severity of seizures. The spasms are characterized by a single or repetitive, flexion and adduction movement of limbs and body. Occasionally these may be extensor or a mixture of both movements. These seizures frequently disappear when the patient is about 3 years of age, but usually mental retardation persists. The EEG pattern is one of pronounced disorganization with high voltage slow waves and multiple spike foci (Figure 2). The bursts of abnormal waves are frequently followed by a short period of decreased electrical activity. Asynchrony and asymmetry are common findings.

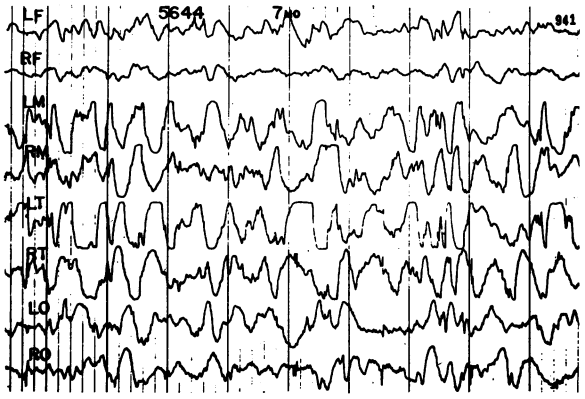


Figure 2.—Disorganization of normal rhythm with diffuse, asynchronous slow waves and spikes.

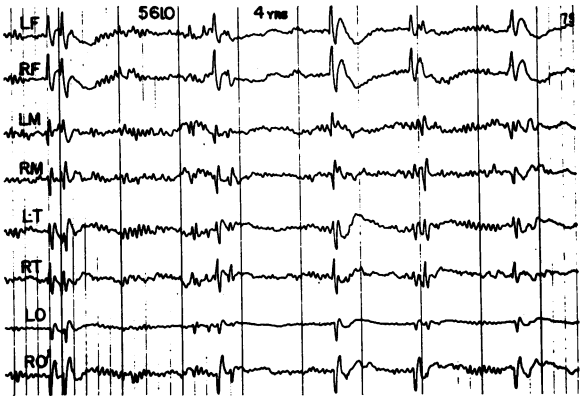


Figure 3.—Diffuse, repetitive spike waves, each followed by suppression of activity. This patient had mental retardation, but without clinical seizures.

In some patients unusual, variable, paroxysmal symptoms—atypical epilepsy—replace overt convulsive movements.[21,30] Pain may be present in any portion of the body, most commonly referred to the head or abdomen. Dizziness or fainting also occur and must be differentiated from the same symptoms due to hypotension. Episodes of unrational rage, laughing or weeping are occasionally noted, as well as autonomic disturbances of sweating, flushing, vomiting and shivering. The most characteristic EEG feature is the appearance of 14 and 6 cps. positive spikes over one or both midtemporal areas. These are frequently only present in the sleeping EEG. Although this finding has been described in some patients with various psychiatric disturbances, including "behavior problems," it should not be construed that it will be present in the case of every nonconformist child.

#### IDIOPATHIC EPILEPSY

The group of conditions included under idiopathic epilepsy are characterized by specific clinical seizure patterns, absence of abnormal neurological findings, high familial incidence and lack of known etiological factors. In instances in which postmortem brain examination has been possible, no anatomic abnormality can be discerned. It is assumed a physiological cerebral defect is present. A brief correlation of clinical and EEG findings follows:

Generalized seizures have neither focal features nor focal aura although sensations of epigastric fullness or a "funny feeling" may be described. The EEG is not specific for this condition and may show a normal interseizure record, or there may be generalized bursts of abnormally fast or slow waves, spikes or sharp waves (Figure 3).

Petit mal is very characteristic with an onset around 4 to 10 years of age, and with the lapses or staring spells of very brief duration. These usually disappear during adolescence. The EEG is characteristic[13]: Normal electrical activity is interrupted by generalized, bilaterally synchronous, 3 per second spike and wave complexes (Figure 4). Hyperventilation is a potent activator of this abnormality. Infrequently the staring spells may be accompanied by myoclonic jerks of muscle groups or the extremities. In these instances the EEG may show the same 3 per second spike and slow waves, but with accompanying multiple spikes.

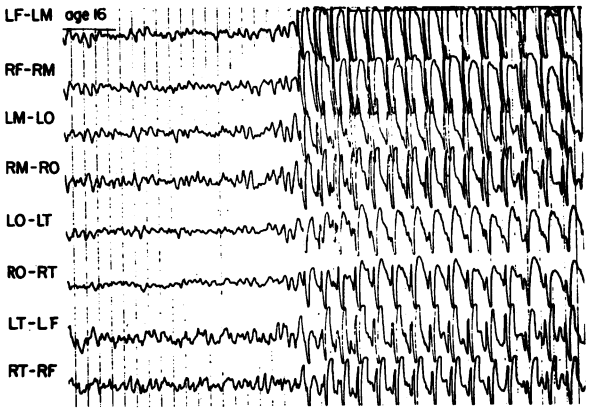


Figure 4.—Showing typical 3 cycles per second spike and slow wave complexes in petit mal, beginning mid-way of tracing.

#### FEBRILE CONVULSIONS

Febrile convulsions constitute a special problem of clinical management and prognosis. In addition to the clinical clues or short duration, generalized seizures developing only with fever, and a positive family history, the EEG will probably be normal in approximately 95 per cent of patients[17,20] who never develop afebrile convulsions. By contrast, a high percentage of patients with initial febrile convulsions, but with abnormal EEG's, will eventually develop afebrile convulsions.

## REPORTING ON EEG EXAMINATION

The EEG report to the attending physician must be clear, succinct and interpretive, but must carefully avoid making a clinical diagnosis or prescribing therapy. If the tracing is sufficiently characteristic, the encephalographer should suggest that it is compatible with a certain kind of pathological process, but should avoid making a clinical diagnosis.

Prognosis of the disease may frequently be confidently outlined. For example, demonstration of the classical 3 per second spike and wave in a patient with lapse attacks suggests a probably excellent long-term outlook. Appearance of the disorganized pattern of hypsarhythmia reflects a very poor outlook for normal mental and motor development.

Evaluation of therapy is also a function of repeated electrical examinations. Disappearance of EEG abnormalities following therapy is good evidence of improvement but the decision regarding modification of therapy must be made by the clinician.

1820 Lincoln Avenue, Anaheim (Carson).

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